Obesity Among Primary School Male Children Aged 10-12 Years in West Gaza City

السمنة لدى الأطفال الذكور (10-12 سنة) في المدارس الابتدائية في غرب مدينة غزة

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بِسْمِ اللَّهِ الرَّحْمَنِ الرَّحِيمِ

(وقَلْ رَبِّ رَزِّنَاهُمْ مِنْ عَلَيْهِمَا)

طَهٌّ ١٤٢١
Dedication

To all the spirit of all Palestinian martyrs who sacrificed themselves to enlighten the way of freedom for us.

To my parents who have always supporting me

To my beloved daughter Aseel

To my brother Ibrahim who helped me to accomplish this thesis.

To my brothers, sister and sons of my sister.

To my university Islamic University-Gaza which is continuously improving the research

To all of them I dedicate this work

ABEER M. SIAM
DECLARATION

I certify that this submission is my own research and that, to the best of my knowledge and belief, it contains material neither previously published or written by another person nor material which to a substantial extent has been accepted for the award of any other degree of the university of other institute, except where due a acknowledgment has been made in the text.

Signature                                         Name                                          Date
Abeer                                     Abeer M. Siam                                    October, 2011

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Abstract

Background: Obesity is a multi-factorial disease that develops from the interaction between genotype and the environment. It is a major public health problem resulting in serious social, physical and psychological damage. The prevalence of obesity and overweight among children is rising to alarming levels in developed and developing countries including Gaza strip.

Objectives: To assess obesity among primary school male children aged 10-12 years in West Gaza City.

Materials and Methods: This case control study comprised 96 obese children (BMI ≥23 kg/m²) and 96 non-obese children (BMI 15-20 kg/m²). Controls and cases were matched in age and socioeconomic conditions. Questionnaire interview was applied. BMI for children was calculated by a computer graphs program specially design to this purpose. Sex, age (years and months), height (in meters) and weight (in kilograms) were introduce into the program. Then by one click on calculate button, the BMI was automatically calculated. Serum leptin, lipid profile and complete blood count were determined. Data were analyzed using SPSS version 18.0.

Results: The prevalence of obesity among primary school male children aged 10-12 years in West Gaza City was 4.3%. Average body mass index of obese children (cases) was 25.8±2.7 whereas that of control children was 17.4±1.0. Family history, eating >3 meals/day, eating while watching television and lack of physical activity in terms of doing exercise and playing football were found to of be factors that affect the obesity among the children. The mean level of leptin was markedly elevated in cases compared to controls (31.5±16.3 Vs 6.9±6.1 ng/ml, % difference=356.5%, P=0.000). When related to BMI, leptin showed positive correlation with BMI (r=0.305 and P=0.003) and total cholesterol (r=0.202 and P=0.049) in obese children. The mean levels of total cholesterol(TC), triglycerides(TAG) and low density lipoprotein (LDL) were significantly increased in cases compared to controls(153.2±34.4 Vs 138.2±22.5 mg/dl, % difference=10.9%, P=0.000; 117.7±34.9 Vs 100.3±27.4
mg/dl, % difference=17.3%, P=0.000; and 84.9±35.9 Vs 69.6±21.1 mg/dl, % difference=22.0, P=0.000, respectively) whereas the mean level of high density lipoprotein (HDL) was significantly decreased in cases (44.2±13.5 Vs 49.5±12.8 mg/dl, % difference=10.7, P=0.000). White blood cells and blood platelets counts were increased in obese children compared to controls. However, the increase in blood platelets was significant (356.2±82.6 Vs 326.5±85.3 X10³cell/μl, % difference=9.1, P=0.014). Red blood cells were significantly increased (4.87±0.44 Vs 4.75±0.42 X10⁵cell/μl, % difference=2.5, P=0.047) whereas MCV and MCH were significantly decreased in obese children (75.9±5.2 Vs 78.4±4.1fl % differences of 3.2, p=0.000 and 24.3±2.3 Vs 25.3±1.9pg % differences of 4.0, p=0.000).

**Keywords:** Obesity, Leptin, Primary school children, Gaza City.
المقدمة:

الخلفية العلمية: السمنة هي مرض متعدد الأسباب ينتج عن تفاعلات التركيب الوراثي والبيئة، وهو مشكلة صحية عامة رئيسية تؤدي إلى إلحاح أضرار اجتماعية ومادية ونفسية، فانشار السمنة وزيادة الوزن بين الأطفال أخذ في الارتفاع إلى مستويات تهدد بالخطر في البلدان المتقدمة والتينامية، كذلك قطاع غزة.

الأهداف: تقييم البدانة لدى الأطفال الذكور في المدارس الابتدائية الذين تتراوح أعمارهم بين 10-12 سنة في غرب مدينة غزة.

المواد والطرفي: تقارن الدراسة الحالة بين 92 طفلاً يعانون من السمنة المفرطة (مؤشر كتلة الجسم لهم ≥ 32 كيلو/متر) و 92 طفلاً غير بداء (مؤشر كتلة الجسم لهم 15-22 كيلو/متر). وتمت تطبيق الأعمار والظروف الاجتماعية والاقتصادية. وتم تعبئة استمانته الخاصة بهم. وتم احصاسب مؤشر كتلة الجسم للأطفال من خلال برنامج صمم حصصًا لهذا الغرض يظهر قياس مؤشر كتلة الجسم على الرسم البياني. وتم تحديد الجنس، واحساب العمر (السنوات والشهر)، والأطوال (بالأمتار) والوزن (بالكيلوغرام). وتم فحص الليbhين، والدهون وخلايا الدم (CBC) للأطفال. وتم تحليل البيانات باستخدام الإصدار SPSS.

النتائج: بلغ معدل انتشار البدانة بين الأطفال الذكور الابتدائية المدارس الذين تتراوح أعمارهم بين 12-10 سنة في غرب مدينة غزة سمنة 4.3% وان مؤشر كتلة الجسم للأطفال البدين (مجموعة التجربة) 25.8 ± 2.7 والصحة المسطحة 17.4 ± 1.0، واتباع متوسط نشاط البدين 3 وحدة/يوم، وتناول الطعام. أثناء مشاهدة التلفزيون وقود النشاط البدني، ورفع كتلة الجسم. ولاحظ ارتفاع في المستوى المتوسط للبدين في المجموعة التجريبية بالمقارنة مع المجموعة الضابطة (P = 0.001، الفرق % = 35.6 ± 6.9). وأظهرت علاقة إيجابية بين البدين مؤشر كتلة الجسم (R=0.202) و (P = 0.001) و (R=0.049) في المجموعة التجريبية و (LDL) في البدين (R=0.202) و (P = 0.001). ووجد زيادة في الكولسترول والدهون الثلاثية و (LDL) في البدين، ووجد زيادة في الكولسترول والدهون الثلاثية و (LDL) في البدين (R=0.202) و (P = 0.001). ووجد زيادة في الكولسترول والدهون الثلاثية و (LDL) في البدين (R=0.202) و (P = 0.001).

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الكلمات الرئيسية : السمنة، اللبتيين، أطفال المدارس الابتدائية في مدينة غزة.
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Chapter 1

Introduction
Chapter 1

Introduction

1.1 Overview

Obesity in general is a multi-factorial disease that develops from the interaction between genotype and the environment. It is a major public health problem resulting in serious social, physical and psychological damage (Mota and Zanesco, 2007). It is measured in terms of a person’s body mass index (BMI) which is determined both by weight and height. BMI cut-off points have been agreed for obese and overweight adults, but for children the situation is more complex. Because a child’s BMI varies with age, different cut-off points have to be used to define overweight and obese children depending on age (Parliamentary Office of Science and Technology, 2003).

The prevalence of obesity and overweight among children is rising to alarming levels in developing and developed countries, (Caius and Benefice, 2002 and Kosti and Panagiotakos, 2006). In the united states, the prevalence of overweight among children and adolescence increased significantly during the 6-year period from 1999 up to 2004 (Ogden et al., 2006). By 2010 it is estimated that 26 million children in European union countries will be overweight including 6.4 who will be obese (Kosti and Panagiotakos, 2006). Epidemic proportions have reached for obesity and overweight among children and adolescents (23.1-30.7%) in Arabian oil-rich countries (El-Bayoumy et al., 2009 and El Mouzan et al., 2010). In the Gaza Strip, there is no available data about the prevalence of obesity and overweight among children.

Many authors proved that there are many determinants of overweight and obesity. Klien-Platate et al. (2003) indicated that overweight among young French adolescents was more frequent in low economic zones and in public schools than in private schools. This was inversely associated with
family income tax. In addition, the high prevalence of obesity and overweight was evident in poor rural communities of Mexico (Sanchez-Castillo et al., 2001). Advanced age, physical inactivity, low economical factors and maternal educational status was reported to be risk factors for obesity among Iranian school children (Mozafari et al., 2007). Other several factors included students behavior, school and family environments may increase overweight risk among low socio-economic status minority students in Chicago (Wang et al., 2007). As a result of a rapid demographic, nutritional and epidemiological transition in Asian countries, dietary habits and life style are changing leading to new health problems. Childhood overweight and obesity are the major example of such problems (Wickramasingh et al., 2004). The main risk factor for obesity in Arab countries generally include feeding habits, lack of physical activity, family income and health education (Ajlouni et al., 1998, Al-Saeed et al., 2006 and Bener, 2006). Systematic review and meta-analysis of published epidemiological studies showed that breastfeeding reduced the risk of obesity in childhood significantly (Arenz et al., 2004 and Scanferla de Siqueiral and Monteirroll, 2007).

Mortality rises exponentially with increasing body weight. The risk of coronary heart disease is doubled in overweight and nearly quadrupled in obese individuals (Willett et al., 1995 and Wilding, 1997). The risk of developing diabetes increases with increasing weight and obese people have a 40 fold higher risk of developing the disease than non-obese people (Al-Nuaim, 1997 and Wilding, 1997). Osteoarthritis and respiratory diseases, particularly sleep apnoea are more common in obese people (Wilding, 1997). Obesity was significantly associated with an increase in both systolic and diastolic blood pressure (Kordy and El-gamal, 1995 and Ashton, 2001), stroke, and certain forms of cancer (World Health Organization, 1998). As obesity is also associated with liver disease, where increasing the risk of elevated liver enzymes by a factor of 2-3, whereas the risk of steatosis on ultrasound is increased by a factor of 3 in the presence of overweight and peaks at a factor of approximately 15 in the presence of obesity (Marchesini et al., 2008).
Some biochemical changes associated with obesity were addressed. Leptin, a dipocyte hormone plays a key role in body weight regulation, is believed to be a potential biomarker for childhood obesity (klein et al.,1998; Venner et al., 2006 and koncsos et al., 2010). Hypercholesterolemia, hypertriglyceridaemia, high level of low density lipoprotein, and low level of high density lipoprotein were found in obese children (Horri and Vakili, 2006; Nascimento et al., 2009 and Yamborisut et at., 2009). In addition, blood cell counts were reported to be associated with body mass index in obese children (kinik et al., 2005 and kelishadi et al., 2010).

Although obesity is prevalent among children in Gaza Strip, the previous studies focused on obesity assessment among adults (Zabut et al., 2007 and AL-jedi, 2011). Therefore, the present work is the first study to assess obesity among primary school male children aged 10-12 years in West Gaza City, Gaza Strip.

1.2 Objectives

The general objective of the current study is to assess obesity among primary school male children aged 10-12 years in West Gaza City. The Specific objectives are:

1. To determine the prevalence of obesity among the primary school male children aged 10-12 years in West Gaza City.

2. To recognize different sociodemographic data in obese and non obese children.

3. To assess feeding, feeding habits and physical activity as risk factors of obesity of the study population.
4. To determine leptin hormone and lipid profile (triacylglycerol, cholesterol, LDL, HDL) in obese and non-obese children.

5. To measure complete blood counts of obese and non-obese children.

6. To verify relations between BMI and the studied parameters.

1.3 Significance and General Objective of the study

In spite of high prevalence of obesity and its risk factors in Gaza Strip there were no available data about obesity prevalence and its determinants among children. However, one study entitled "Leptin and soluble leptin receptor among obese adults in the Gaza Strip" was recently published (Zabut et al., 2007). In addition, there are no previous studies speculate the role of leptin in obesity among children in the Gaza Strip. Therefore, the present study is the first to assess obesity among primary school male children aged 10-12 years in West Gaza City.
Chapter 2

Literature Review
Chapter 2

Literature Review

2.1 Definition of obesity

Obesity is a condition in which excess body fat has accumulated to an extent that health may be negatively affected. Obesity is the consequence of an overall positive energy balance maintained over time, that is, the metabolizable energy intake exceeds the energy expenditure for basal metabolic requirements, thermoregulation, physical activity, and growth (Rosenbaum et al., 1997).

2.2 Assessment and classification of obesity

Obesity is measured in terms of a person’s body mass index which is determined both by weight and height. Body mass index (BMI) of children differs from adult, the assessment of weight status is much more complex. This is because children are growing and the growth patterns (and hence the BMI) of children differs by age (Parliamentary Office of Science and Technology, 2003 and Barbara and Clyde Park., 2009). The pattern of growth is dependent upon the sex of the child since the growth pattern for boys is very different from the growth pattern for girls. For children, BMI is determined — based on carefully measured height and weight — and then graphs or growth charts are used to find each child’s BMI percentile-for-age by plotting the BMI value versus age on a growth chart for that child’s sex. To use a BMI chart, you must know the birth date of the child, so that you can calculate the age of the child (in months) on the date that his or her height and weight were measured. The BMI graphs or growth charts were developed by Centers for Disease Control and Prevention (2000). Accordingly, children BMI (BMI for age percentile, age 2-19 years) was classified as follows: Underweight ≤ 5 th percentile, healthy weight 5 th to the 85 th percentile, overweight 85 th to the 95 th percentile and obese ≥95 th percentile (Benjamin,
2010). In the present study children aged 10-12 years and the normal BMI was calculated at 15.0-20.0 k/m$^2$ whereas the BMI for obese children was calculated to be ≥23.0 k/m$^2$.

2.3 Prevalence of obesity

The prevalence of obesity has reached alarming levels, affecting virtually both developing and developed countries of all socio-economic groups, irrespective of age, sex or ethnicity. Concerning childhood obesity, the prevalence of overweight in Africa and Asia averaging well below 10% and in the Americas and Europe above 20% (Kosti and Panagiotakos, 2006). In the USA, 25% of children are overweight and 11% are obese (Dehghan et al., 2005).

In retrospective series of cross sectional studies of routinely collected data, Bundred et al. (2001) determined trends in weight, height, and BMI in 64430 children between 1989 and 1998 in United Kingdom. From 1989 to 1998 there was a highly significant increasing trend in the proportion of overweight children (14.7% to 23.6%; P < 0.001) and obese children (5.4% to 9.2%; P < 0.001).

Wang et al. (2002) examined the trends of overweight and underweight in young persons aged 6–18 years from the United States, Brazil, China, and Russia using international references. Nationally representative data from Brazil (1975 and 1997), Russia (1992 and 1998), and the United States (1971–1974 and 1988–1994) and nationwide survey data from China (1991 and 1997) were used. The prevalence of overweight increased during the study periods in Brazil (from 4.1 to 13.9), China (from 6.4 to 7.7), and the United States (from 15.4 to 25.6); underweight decreased in Brazil (from 14.8 to 8.6), China (from 14.5 to 13.1), and the United States (from 5.1 to 3.3). In Russia, overweight decreased (from 15.6 to 9.0) and underweight increased (from 6.9 to 8.1). The annual rates of increase in the prevalence of overweight were 0.5% (Brazil), 0.2% (China), -1.1% (Russia), and 0.6% (United States).
The prevalence of overweight and obesity in Saudi children with ages ranging up to 18 years was reviewed (El-Hazmi and Warsy, 2002). The study was a cross-sectional national epidemiological household survey, and the study group included 12701 children (boys 6281; girls 6420). Their height and weight were measured and body mass index (BMI) was calculated. The overall prevalence of overweight was 10.7% and 12.7% in the boys and girls, respectively, and obesity was 6.0% and 6.74% in the two groups, respectively. The children were grouped according to the province to which they belonged, and prevalence of obesity and overweight were calculated for each province. The highest frequency was in the Eastern Province, while the lowest was in the Southern Province. In Eastern Mediterranean Region a prevalence of 3%–9% overweight and obesity has been recorded among preschool children, while that among schoolchildren was 12%–25% (Musaiger, 2004).

In a descriptive cross-sectional study, Abu Baker and Daradkeh (2010) determined the prevalence of overweight and obesity among adolescents aged 13–16 years in Irbid governorate, Jordan, and compared the prevalence by sex, residential area and socioeconomic status. In a cluster random sample of 1355 school students the prevalence of overweight and obesity (body mass index ≥ 85th percentile) was 24.4% (15.7% overweight and 8.7 % obese) and was significantly higher among female students, students who lived in urban areas and those with working parents.

El-Bayoumy et al. (2009) found out the prevalence of obesity and overweight among intermediate school Kuwaiti adolescents aged 10 to 14 years. The study comprised a multistage stratified random sample that included 5402 children (2657 males and 2745 females). The weights and heights were measured, from which the body mass index (BMI) was calculated. BMI values higher than 95 percentile were accepted as being obese and those in between 85 and 94 percentile were accepted as overweight. The overall prevalence of overweight and obesity was 30.7% and 14.6%, respectively. The overall prevalence of overweight and obesity among males was 29.3% and 14.9%, respectively (P < .001) and the prevalence of
overweight and obesity among females was 32.1% and 14.2%, respectively (P < 0.001).

The correlation of the prevalence of overweight and obesity among 460 primary school children aged 8–11 years in Aydin, Turkey with certain sociodemographic characteristics and the prevalence of overweight and obesity in the children's families were determined (Alışır and Karaçam, 2011). Study data were collected using a questionnaire. The prevalence of overweight and obesity was found to be 12.8% and 13.7%, respectively and this condition was correlated with the number of family members and the parents' body mass index.

### 2.4 Sociodemographic factors and obesity

Obesity is a multi-factorial disease that develops from the interaction between genotype and the environment. Polymorphisms in various genes controlling appetite and metabolism predispose to obesity when sufficient food energy present. As of 2006 more than 41 of these sites have been linked to the development of obesity when a favorable environment is present (Poirier et al. 2006). Initial reports indicated that although obesity is associated with higher socioeconomic status in early childhood, it begins to predominate in poorer females in adolescence. In general, excess weight gain in children was reported to be more prevalent among lower income families (Wang et al., 2002).

Alaimo et al. (2001) investigated associations between family income, food insufficiency, and being overweight in US children aged 2 to 7 and 8 to 16 years. Among older non-Hispanic white children, children in families with low income were significantly more likely to be overweight than children in families with high income. There were no significant differences by family income for younger non-Hispanic white children, non- Hispanic black children, or Mexican American children. After adjusting for confounding variables, there were no differences in overweight by food sufficiency status, except that younger food-insufficient girls were less likely to be overweight, and non-
Hispanic white older food insufficient girls were more likely to be overweight than food-sufficient girls (P <0.10).

The associations between socioeconomic status (SES), two levels of subjective social status (SSS), and obesity of 1491 black and white adolescents attending public school in Ohio were characterized (Goodman et al., 2003). Although there were no sex differences in SES, black students were more likely to come from families with less well-educated parents and lower incomes (P<0.001). Black girls had the lowest societal SSS (P = 0.003), lowest school SSS (P=0.046), and highest BMI (P<0.001). Prevalence of overweight was highest among black girls (26.0%) and boys (26.2%), intermediate for white boys (17.2%), and least for white girls (11.6%). Logistic regression modeling revealed that parent education, household income, and school SSS were each associated with overweight.

Lamerz et al. (2005) assessed the association between socioeconomic status (SES) and childhood obesity among 2020 German children and which factor in particular stands out in relation to obesity. The indicators of parental education were most strongly associated with children's obesity. There was a strong dose–response relationship between a composed index of social class and obesity. Children of the lowest social status had a more than three-fold risk to be obese than children of the highest social status in the screening population (OR: 3.29, CI: 1.92–5.63).

The association between parental obesity and childhood obesity was studied (Kumar et al., 2010). Children in middle schools were screened for obesity. For each obese child two controls were studied. Parental history of obesity was present for 32.7% of obese children. Children with parental history of obesity showed 25.2 times more chances of developing obesity than controls. The percentage of 33.8 of the obese girls and 31.6% of the obese boys had history of parental obesity. If the father was obese, boys had 6.5 times more chance and girls had 40.1 times more chance of developing obesity. Mother’s obesity had an influence on 23.7 % of the boys and only 16 % of the girls.
The prevalence of overweight and obesity in primary schoolchildren and its correlation with sociodemographic factors in Aydin, Turkey was determined (Alishir and Karaçam, 2011). The study was done with 460 children aged 8–11 years. Study data were collected using a questionnaire. The study determined the children's overweight prevalence as 12.8% and their obesity prevalence as 13.7%. It was found that having a family with four or fewer members increased the risk of overweight and obesity (OR = 1.889; 95% CI = 1.086–3.287), that the mother's education level of secondary school or less decreased the risk of overweight and obesity of children (OR = 0.458; CI = 0.268–0.780), and that the greater the mother's (OR = 1.069; CI = 1.008–1.133) and father's (OR = 1.127; CI = 1.049–1.210) body mass index, the more pronounced was the children's risk of being overweight.

2.5 Feeding and feeding habits

von Kries et al. (1999) assessed the impact of breast feeding on the risk of obesity and risk of being overweight in 13345 German children at the time of entry to school. Early feeding, diet, and lifestyle factors were assessed using responses to a questionnaire completed by parents. The prevalence of obesity in children who had never been breast fed was 4.5% as compared with 2.8% in breastfed children. A clear dose-response effect was identified for the duration of breast feeding on the prevalence of obesity: the prevalence was 3.8% for 2 months of exclusive breast feeding, 2.3% for 3-5 months, 1.7% for 6-12 months, and 0.8% for more than 12 months. Similar relations were found with the prevalence of being overweight. The protective effect of breast feeding was not attributable to differences in social class or lifestyle. After adjusting for potential confounding factors, breast feeding remained a significant protective factor against the development of obesity (odds ratio 0.75, 95% CI 0.57 to 0.98) and being overweight (0.79, 0.68 to 0.93).

Gillman et al. (2001) examined the extent to which overweight status among 8186 girls and 7155 boys, aged 9 to 14 years is associated with the type of infant feeding (breast milk vs infant formula) and duration of breastfeeding. Data were collected through a questionnaire. In the first 6
months of life, 9553 subjects (62%) were only or mostly fed breast milk, and 4744 (31%) were only or mostly fed infant formula. A total of 7186 subjects (48%) were breastfed for at least 7 months while 4613 (31%) were breastfed for 3 months or less. At ages 9 to 14 years, 404 girls (5%) and 635 boys (9%) were overweight. Among subjects who had been only or mostly fed breast milk, compared with those only or mostly fed formula, the odds ratio (OR) for being overweight was 0.78 (95% confidence interval [CI], 0.66-0.91), after adjustment for age, sex, energy intake, time watching television, physical activity and mother’s body mass index. Compared with subjects who had been breastfed for 3 months or less, those who had been breastfed for at least 7 months had an adjusted OR for being overweight of 0.80 (95% CI, 0.67-0.96).

Breastfeeding was suggested to reduce the risk of child obesity to a moderate extent. Of 11 studies that examined prevalence of overweight in children older than 3 years of age and that had a sample size of ≥ 100 per feeding group, 8 showed a lower risk of overweight in children who had been breastfed, after controlling for potential confounders. If the association is causal, the effect of breastfeeding is probably small compared to other factors that influence child obesity, such as parental overweight (Dewey, 2003).

The rising prevalence of obesity in children in relation to the consumption of sugar-sweetened drinks was examined (Ludwig et al., 2001). Five hundred and forty eight schoolchildren aged 11.7±0.8 years from public schools in four Massachusetts communities were enrolled. For each additional serving of sugar-sweetened drink consumed, both body mass index (mean 0.24 kg/m²; 95% CI 0.10-0.39; P=0.03) and frequency of obesity (odds ratio 1.60; 95% CI 1.14-2.24; P=0.02) increased after adjustment for anthropometric, demographic, dietary, and lifestyle variables. Baseline consumption of sugar-sweetened drinks was also independently associated with change in body mass index (mean 0.18 kg/m² for each daily serving; 95% CI 0.09-0.27; P=0.02). James and Kerr (2005) found that children who consume sugar-sweetened drinks have a higher energy intake and are more likely to become overweight.
Scanferla de Siqueira and Monteiro (2007) examined the association between breastfeeding and obesity in 555 school children aged 6-14 years from Brazilian families of high socioeconomic status. Obesity – the outcome variable – was defined as body mass index at or above the 85th centile plus sub scapular and triceps skin folds at or above the 90th centile using the sex and age specific standards of the US National Center for Health Statistics. Prevalence of obesity in the studied population was 26%. After confounder adjustment, the risk of obesity in children that had never been breastfed was twice that of other children (OR=2.06; 95% CI: 1.02; 4.16). There was no dose response effect of duration of breastfeeding on prevalence of child obesity.

Regarding TV/video viewing, Vandewater et al. (2004) indicated that children with higher weight status spent more time in sedentary activities e.g. watching TV and playing video games than those with lower weight status. In addition, Dennison et al. (2002) described the TV/video viewing habits of a multiethnic, low-income children and determined whether TV/video viewing is related to their adiposity. Mean TV/video viewing times were higher among black children and Hispanic children than white children and increased with the child’s age. In multiple logistic regression, the odds ratio of children having a BMI >85th percentile was 1.06 (95% CI: 1.004-1.11) for each additional hour per day of TV/ video viewed, independent of child age, child sex, parental educational attainment, and race/ethnicity. Almost 40% of children had a TV set in their bedroom; they were more likely to be overweight and spent more time (4.6 h/week) watching TV/video than children without a TV in their bedroom. The odds ratio of having a BMI >85th percentile was 1.31 (95% CI: 1.01-1.69) among those with a TV in their bedroom versus those without a TV, after statistical adjustment for child age, child sex, child TV/video viewing h/week, maternal BMI, maternal education, and race/ethnicity.

2.6 Physical activity

most available evidence suggests that a lower activity-related energy expenditure is an important contributor to the increasing prevalence of
obesity, although a blunted response to food intake and reductions in resting energy expenditure may have an impact on weight gain (Albo et al., 1997 and Weinsier et al., 1998). Furthermore, studies have often found associations between leisure-time physical activity (inverse) or total amount of time spent sitting down (direct) and BMI (Martinez-González et al., 1999). A low participation in sports activities, a lack of interest in taking exercise and a high number of hours spent sitting down at work are statistically significant predictors of obesity (Martinez et al., 1999).

Trost et al. (2001) compared the physical activity patterns and the hypothesized psychosocial and environmental determinants of physical activity in an ethnically diverse sample of 54 obese and 133 non-obese sixth grade USA children (mean age of 11.4±0.6). Physical activity measurements were collected over a 7-day period. Compared to their non-obese counterparts, obese children exhibited significantly lower daily accumulations of total counts, moderate physical activity and vigorous physical activity as well as significantly fewer 5, 10 and 20 min bouts of moderate-to-vigorous physical activity. Obese children reported significantly lower levels of physical activity self-efficacy, were involved in significantly fewer community organizations promoting physical activity and were significantly less likely to report their father or male guardian as physically active.

The total daily energy expenditure and physical activity pattern of 18 obese children aged 6-17 years and 18 age- and sex-matched non-obese Hong Kong children were investigated (Yu et al., 2002). The total daily energy expenditure of the obese children was significantly lower (by 22%) than that of non-obese children. Obese children spent 12% less time asleep, but 51% more time in sedentary activity and 30% less time physically active: a ratio of active-to-sedentary waking time of 0.6 for obese children and 1.9 for non-obese children.

Page et al. (2005) determined the levels and patterns of physical activity in a sample of obese and non-obese children in United Kingdom. In all 11 (16.9%) of the 65 girls and 14 (20.6%) of the 68 boys were classified as
obese. Children were instructed in the use of the accelerometer either while at school or in the clinic, and wore the instrument while carrying out their normal daily activities for 7 days. Obese children were significantly less physically active overall than their non-obese counterparts (31844±13 200 vs 41844±10 430 counts/h; 95% confidence interval 4407 to 15592; P=0.001). Similarly the obese children spent less time in physical activity of moderate or greater intensity than the non-obese children (9.9±3.9 vs 12.9±4.2 min/h; 95% confidence interval 1.15 to 4.80; P=0.002). Hourly patterns of activity indicated a tendency in obese children to be less active than non-obese children at times when activity was more likely to be determined by free choice, particularly outside of school time.

Lioret et al. (2007) assessed the prevalence of childhood overweight and obesity in French children aged 3–14 years and examined how physical activity and sedentary behavior are involved in the association between socioeconomic status and overweight, while taking into account total energy intake. In total, 15.2% of the children are overweight including obese. Overweight is inversely associated with socioeconomic status in children over 6 years of age. Leisure-time physical activity is negatively correlated to overweight among the 3 to 5-year-old children only, whereas sedentary behavior is positively related to overweight in childhood and adolescence. High daily caloric intake by the obese and overweight children and physical inactivity was reported among the majority of them (El-Bayoumy et al., 2009).

The role of physical activity programs and recess for a national cohort from first grade to fifth grade 8246 USA children in 970 schools on body mass trajectories was assessed (Fernandes and Sturm, 2011). Among first graders, 7.0% met the National Association of Sport and Physical Education (NASPE) recommended time for PE and 70.7% met the recommended time for recess in the previous week. Boys experienced a greater increase in body mass than girls. Meeting the NASPE recommended time for recess was associated with a 0.74 unit decrease in BMI percentile for children overall. Meeting the NASPE recommendation for physical education was associated with 1.56 unit decrease in BMI percentile among boys but not girls. This
indicates that meeting the national recommendations for PE and recess is effective in mitigating body mass increase among children.

2.7 Biochemical and hematological parameters

Leptin was identified through positional cloning of the obese (ob) gene, which is mutated in the massively obese ob/ob mouse, and it has a pivotal role in regulating food intake and energy expenditure (Zhang, 1994). Leptin is a small peptide hormone (16-kDa Protein) that is mainly produced in adipose tissue (Kiess et al., 1998). Although leptin is a circulating signal that reduces appetite, in general, obese people have an unusually high circulating concentration of leptin (Considine et al., 1996). These people are resistant to the effects of leptin. Leptin could be regulated by insulin (Susan, 2000). The high sustained concentrations of leptin form the enlarged adipose stores result in leptin desensitization. The pathway of leptin control in obese people might be flawed at some point so the body doesn't adequately receive that satiety feeling subsequently to eating (Considine et al., 1996). In addition, obese individuals showed higher serum lipid profiles than non-obese ones (Garcés et al., 2005 and Yamborisut et al., 2009).

Lahlou et al. (1997) assessed circulating leptin in normal children and during the dynamic phase of juvenile obesity its relation to body fatness, energy metabolism, caloric intake, and sexual dimorphism. In 112 obese compared with 42 lean children, serum leptin was elevated early in the evolution of childhood-onset obesity (28.4±1.4 vs. 4.5±0.4 ng/ml in lean children, P < 0.0001) and correlates with adiposity. Obese children also had higher serum leptin normalized to fat mass. Despite high serum leptin, obese children ingested 2-3 times more calories than did lean control subjects (P < 0.0001) and gained weight rapidly (10.2±0.3 vs. 2.9±0.1 kg/year in control subjects, P < 0.0001). Girls had higher leptin levels than did boys, in obese as well as in non-obese children, and showed a closer correlation between adiposity and serum leptin. Elevation of serum leptin was comparable before and after puberty in obese boys, but puberty further increased leptin levels in
obese girls (36±3 ng/ml), resulting in a clear sexual dimorphism with pubertal obese boys (22±5 ng/ml, P < 0.005).

The biochemical changes in obese children in Taiwan, in comparison with those observed by other authors were investigated (Hwang et al., 2001). Children with weights above 120 percent of ideal body weight were considered obese. Serum glucose, insulin, uric acid, and creatinine levels and lipid profiles of 298 obese children (mean age 11.3±2.4 years, 158 boys) and 60 controls (mean age 11.6±2.2 years, 38 boys) were determined using accepted procedures. Demographic and serum biochemical characteristics were compared between obese and control groups overall and by gender. Serum glucose, insulin, cholesterol, and triglyceride levels were higher in the obese children than in the control group.

Garce´s et al. (2005) established the relationship between obesity and its metabolic consequences from a total of 1048 Spanish schoolchildren, 6 to 8 years of age. The prevalence of obesity and overweight were 9.4% and 15.7%, respectively, in boys and 10.5% and 18.0%, respectively, in girls. In both sexes, obese children had higher triglycerides and lower high-density lipoprotein-cholesterol levels than non-obese children. No differences were found in plasma glucose or low-density lipoprotein-cholesterol levels between normal and obese children. However, insulin levels and the homeostasis model assessment for insulin resistance were significantly (P<0.001) higher in obese children of both sexes but that free fatty acid levels were lower in obese children than in nonobese children, with a statistical significance in girls (0.72±0.30 vs 0.61±0.16 mEq/liter).

Complete blood count (CBC) parameters and peripheral blood CD34(+) cell counts in prepubertal obese and nonobese children were compared (Klinik et al., 2005). Relationships between leptin levels and CBC parameters and peripheral CD34(+) progenitor cell counts in the obese group were also investigated. Thirty one healthy, prepubertal, obese children and 30 nonobese, age-matched prepubertal controls were included in the study. In the obese group, the mean results for body mass index (BMI), BMI standard
deviation score (BMI SDS), and serum leptin level were significantly higher than the corresponding control findings. There were no significant differences between the groups with respect to CBC parameters and CD34(+) cell percentage. In both the obese and control groups, the girls' serum leptin levels were significantly higher than the boys. In the obese group, serum leptin level was strongly correlated with BMI and with BMI SDS.

Ustundag et al. (2006) evaluated the oxidative and antioxidant status and any correlation with leptin in obese prepubertal children. A cross-sectional study was made of healthy children from ten elementary schools in Eastern Turkey. Blood samples were drawn from children comprising obese and control groups, on a visit to their school in the morning after an overnight fast. The mean body mass index (BMI) was 24.03 ± 4.09 kg/m² in the obese group and was 17.51 ± 2.33 kg/m² in the control group. Mean plasma leptin concentration was significantly higher in the obese children. Homocysteine and malondialdehyde (MDA) levels were also significantly higher in the obese group. In contrast superoxide dismutase (SOD) and glutathione peroxidase activities were significantly decreased in the obese group (p < 0.001).

Serum leptin levels in obese Indian children and its correlation to anthropometric and biochemical parameters were evaluated (Dubey et al., 2007). Leptin levels were measured in 36 children (26 boys, age 1.5 to 15 years) and 37 adults (21 men, age 25 to 69 years) with obesity and 29 normal weight controls (15 children and 14 adults). Leptin levels were higher than controls in obese children (19.4 ± 6.4 ng/mL against 5.4 ± 1.7 ng/mL, p = 0.0001) and obese adults (18.9 ± 6.4 ng/mL against 7.8 ± 5.6 ng/mL, p = 0.0001). Leptin levels were higher than males in obese girls (23.5 ± 1.7 ng/mL against 18.0 ± 7.6 ng/mL, p = 0.040) and women (21.3 ± 4.4 ng/mL against 15.8 ± 7.4 ng/mL). Leptin levels correlated with body mass index, waist circumference and waist to-hip ratio. A positive correlation was observed between serum leptin and cholesterol, triglycerides and LDL-cholesterol. No correlation was seen with fasting blood glucose and HDL-cholesterol.
Ghergerehchi (2009) evaluated the frequency and patterns of dyslipidemia in overweight and obese children and determined the extent of blood lipid abnormality in overweight and obese children. A prospective matched case control study on 230 overweight and obese children and adolescents aged 4 to 18 years was undertaken. The control group included 50 nonobese children. The total frequency of dyslipidemia was 69.58%. The prevalence of dyslipidemia increased with severity of obesity and reached 76.9% in the severely obese (P<0.005). High triglycerides was the most common dyslipidemia in combination (26.08%) and in isolation (18.6%). There was a significant difference in mean of triglycerides between the severely obese and other groups (P<0.004).

The relationship between serum leptin concentrations and body composition among a sample of obese Thai children was investigated (Yamborisut et al., 2009). A cross-sectional study was conducted in 158 schoolchildren, of whom 107 were obese and 51 normal weight; their mean age was 8.2 years. Results revealed subcutaneous fat skin fold, total body fat and waist circumference (WC) were significantly higher in obese than normal weight children (P<0.001). Serum leptin levels and lipid profile results i.e. serum triglycerides (TG), serum total cholesterol (TC), low density lipoprotein cholesterol (LDL-C) and energy intake, were also significantly higher in the obese children than their normal-weight peers. Stepwise multiple regression analysis indicated that among boys, WC (P<0.001) and serum TG (P=0.019), and among girls, WC (P<0.001) and TBF (P=0.030), were significantly associated with leptin concentrations. No associations were found between leptin and energy intake in these children.

Kelishadi et al. (2010) determined the association of complete blood count with obesity and cardiometabolic risk factors in children. The study comprised 326 (172 girls and 154 boys) obese Iranian children aged 6-12 years. A significant increasing trend in the mean body mass index (BMI), waist circumference (WC), triglycerides (TG), total- and low density lipoprotein (LDL)- cholesterol were documented across the quartiles of the white blood cell (WBC) count, and for waist-to-hip ratio and total cholesterol across
platelet quartiles. A similar increasing trend was documented for BMI, waist and hip circumference, diastolic blood pressure, LDL-C, and for TG from the second to the fourth quartile of the red blood cells. By the increase in the number of components of metabolic syndrome, the mean BMI, WBC, and TG increased significantly. The highest correlation was documented between WBC count and TG. The WBC count increased the risk of increased BMI (odds ratio [OR]=1.45, confidence interval [CI] 95%; 1.11-1.65, p=0.001), increased WC (OR; 1.47, CI 95%; 1.15-1.74, p=0.001), and high TG (CI 95%; 1.241.06-1.44, p=0.005 ).
Chapter 3

Materials and methods
Chapter 3

Materials and Methods

3.1 Study design

The present study is a case control.

3.2 Study population

The study population included the primary school male obese children (BMI $\geq 23.0 \text{ kg/m}^2$) in West Gaza City aged 10-12 years old. The controls were non-obese children with BMI 15-20 kg/m$^2$.

3.3 Sample size

It comprised all male obese children (n=96 and BMI $\geq 23\text{ kg/m}^2$) from different classes of the 13 primary schools in the West Gaza City. A total of 96 normal weight control male children (BMI=15-20kg/m$^2$) were selected from the same classes. Controls and cases were matched in age and socioeconomic conditions.

3.4 Questionnaire

A meeting interview was used for filling in the questionnaire for both cases and controls (Annex 1). All interviews were conducted face to face with children parents by the researcher himself. During the survey the interviewer explained any of the questions that were not clear. Most questions were of one of two types: the yes/no question, which offer a dichotomous choice; and the multiple choice questions, which offer several fixed alternatives (Backestrom and Hursh-Cesar, 1981). The questions were direct and brief and the validity of the questionnaire was tested by six specialists in the fields
of epidemiology, public health, biochemistry and nutrition. The questionnaire included personal information, sociodemographic data (education and occupation of the children parents, family income/month and family history of obesity), feeding and feeding habits (breast feeding, drinking soft drink, eating sweets, eating indomy, number of meals/day and eating while watching television) and physical activity (doing exercise and playing football) and breast feeding and family health education. Pilot study was done prior to beginning real data collection to know the length and clarity of questionnaire and to evaluate the outcome. Twelve individuals were interviewed. At the end of the pilot study, a comprehensive revision to questionnaire was made and modified as necessary. The pilot subjects were not included in the study.

3.5 Eligibility

3.5.1 Inclusion criteria

A. Case group
Obese children (BMI ≥ 23kg/m²) aged 10-12 years old.

B. Control group
Normal weight children (BMI 15-20kg/m²) aged 10-12 years old.

3.5.2 exclusion criteria
Apparently not healthy children were excluded

3.6 Ethical consideration

An approval to carry out the study was obtained from the Helsinki Committee-Gaza (annex 2). One official letter of requests was sent to Ministry of Education and Higher Education to obtain approval to conduct the study in the Government schools of West Gaza City (annex 3). Parents of the children were given a full explanation about the purpose of the study assurance about the confidentiality of the information and that the participation was optional.
3.7 Body mass index (BMI)

BMI was used for evaluation the obesity. To measure BMI, one begins by weighting the child in light clothes and without shoes. Height is measured without shoes by using a pediatric measuring board. Medical balance (seca model 762, Germany) was used for this purpose. BMI for children was calculated by a computer graphs program specially design for this purpose (Centers for Disease Control and Prevention, 2000). The following parameters were introduce into this program: sex, age (years and months), height (in meters) and weight (in kilograms). Then by one click on calculate button, the BMI was automatically calculated. Body mass index: Normal=15.0-20.0, Obese=$\geq$23.0 kg/m$^2$.

3.8 Blood sample collection and processing

Venous blood sample (about 7 ml) was drawn by a well trained medical technologist into vacutainer tubes from overnight fasting the case and the control children. About 2 ml blood was placed into EDTA vacutainer tube for complete blood count. The remainder quantity of blood was left for a while without anticoagulant to allow blood to clot. Then serum samples were obtained by centrifugation at room temperature by Rotina 46 Hettich centrifuge, Japan at 4000 rpm/10 minutes and then samples were stored in refrigerator until biochemical analysis.

3.9 Biochemical analysis

3.9.1 Determination of serum leptin

Determination of human serum leptin level was carried out by competitive enzyme immunoassay (Diagnostic System Laboratories (DSL), USA) technique.
Principle
The DSL-10-23100 ACTIVE Human Leptin ELISA is an enzymatically amplified "two step" sandwich-type immunoassay. In the assay, Standards, Controls and unknown serum or plasma samples were incubated in microtiteration wells, which have been coated with anti-human leptin antibody. After incubation and washing, the wells were treated with another anti-human leptin detection antibody labeled with the enzyme horseradish peroxidase (HRP). After a second incubation and washing step, the wells were incubated with the substrate tetramethylbenzidine (TMB). An acidic stopping solution was then added and the degree of enzymatic turnover of the substrate was determined by dual wavelength absorbance measurement at 450 and 620 nm. The absorbance measured was directly proportional to the concentration of human leptin present. A set of human leptin standards was used to plot a standard 35 curve of absorbance versus human leptin concentration from which the human leptin concentrations in the sample can be calculated.

Assay procedure
Annabel all specimens and reagents to reach room temperature (~25°C) and mix thoroughly by gentle inversion before use. Standards, Controls and samples should be assayed in duplicate.
1. The microtiteration strips were marked to be used.
2. Twenty five microliters of the standards, controls and samples were pipeted into the appropriate wells.
3. One hundred microliters of the Assay Buffer E were added to each well using a semi-automatic dispenser.
4. Incubate the wells, shaking at a fast speed (500-700 rpm) on an orbital microplate shaker, at room temperature (~25 °C) for 2 hours.
5. Aspirate and wash each well 5 times with the Wash Solution using an automatic microplate washer. Blot dry by inverting plate on absorbent material.
6. The Antibody-Enzyme Conjugate Solution was prepared by diluting the Antibody-Enzyme Conjugate Concentrate in the Assay Buffer.

7. One hundred microliters of the Antibody-Enzyme Conjugate Solution was added to each well using a semi-automatic dispenser.

8. The wells were incubated, shaked at a fast speed (500-700 rpm) on an orbital microplate shaker, at room temperature (~25 °C) for 1 hour.

9. Aspirate and wash each well 5 times with the Wash Solution using an automatic microplate washer. Blot dry by inverting plate on absorbent material.

10. One hundred microliters of the TMB Chromogen Solution was added to each well using a semi-automatic dispenser.

11. Incubate the wells, shaking at a fast speed (500-700 rpm) on an orbital microplate shaker, at room temperature (~25°C) for 10 minutes. Avoid exposure to direct sunlight.

12. One hundred microliters of the Stopping Solution (0.2M sulfuric acid) was added to each well using a semi-automatic dispenser.

13. The absorbance of the solution in the wells was read within 30 minutes, using a microplate reader set to 450 nm.

**Calculation**

A. The mean absorbance for each standard, control and samples were calculated.

B. Plot the log of the human leptin concentrations in ng/mL along the x-axis versus the mean absorbance readings for each of the standards along the y-axis versus, using a linear curve-fit. Alternatively, the data can be plotted linear vs. linear and a smoothed spine curve-fit can be used.

C. Determine the human leptin concentrations of the controls and samples from the standard curve by matching their mean absorbance readings with the corresponding human leptin concentrations.
3.9.2 Determination of serum total cholesterol

Enzymatic colorimetric method for the quantitative determination of total Cholesterol in serum or plasma, using Globe diagnostics kit, Italy.

Indication
Cholesterol determination is used for the diagnosis and monitoring of lipidic metabolism diseases.

Principle
The measurement is based on the following enzymatic reactions:

\[
\text{CHE} \\
\text{Cholesterol esters} + \text{H}_2\text{O} \rightarrow \text{Cholesterol} + \text{Fatty acids}
\]

\[
\text{CHOD} \\
\text{Cholesterol} + \text{O}_2 \rightarrow \text{Cholest-4-en-3-one} + \text{H}_2\text{O}_2
\]

\[
\text{POD} \\
2\text{H}_2\text{O}_2 + \text{hydroxybenzoate} + 4\text{-Amminoantipyrine} \rightarrow \text{Red complex} + 4\text{H}_2\text{O}
\]

The intensity of the red complex is proportional to the total cholesterol present in the sample.

Assay procedure
About 0.5 ml of serum was transferred to the Mindray BS-120 chemistry autoanalyzer to perform the test according to these parameters:

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Reagent (µl)</td>
<td>300</td>
</tr>
<tr>
<td>Serum (µl)</td>
<td>3</td>
</tr>
<tr>
<td>Incubation period (s)</td>
<td>17×18</td>
</tr>
<tr>
<td>Reaction type</td>
<td>End point</td>
</tr>
<tr>
<td>Wavelength (nm)</td>
<td>510</td>
</tr>
</tbody>
</table>
Reference range

Cholesterol values according to a study on a population of adults in absence of coronary disease are the following:

<table>
<thead>
<tr>
<th>Recommended values</th>
<th>&lt; 200 mg/dl</th>
</tr>
</thead>
<tbody>
<tr>
<td>Upper limit</td>
<td>200 - 239 mg/dl</td>
</tr>
<tr>
<td>High value</td>
<td>≥ 240 mg/dl</td>
</tr>
</tbody>
</table>

3.9.3 Determination of serum triglyceride

Enzymatic colorimetric method for the quantitative determination of triglycerides in serum or plasma, using Globe diagnostics kit, Italy.

Indication

Triglycerides determination is used for the diagnosis and monitoring of lipidic dysfunction for the evaluation risk of the atherosclerotic disease. Recent studies have demonstrated that high levels of triglycerides, accompanied to an increase of low density lipoproteins (LDL), constitute a particular elevated risk for "coronary heart disease" (CHD).

Principle

Glycerol, released from triglycerides after hydrolysis with lipoproteinlipase is transformed by glycerolkinase into glycerol-3-phosphate which is oxidized by glycerolphosphate oxidase into dihydroxyacetone phosphate and hydrogen peroxide. In presence of peroxidase, the hydrogen peroxide oxidizes the chromogen ESPT(4-aminophenazone/N-ethylmethyilanilin- propan-sulphonate sodic) to form purple quinoneimine whose colour intensity, measured at 510 nm, is proportional to the concentration of triglycerides in the sample.

\[
\text{LPL} \\
\text{Triglycerides} \rightarrow \text{Glycerol + Fatty acids}
\]
Assay procedure

About 0.5 ml of serum was transferred to the Mindray BS-120 chemistry autoanalyzer to perform the test according to these parameters:

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Reagent (µl)</td>
<td>300</td>
</tr>
<tr>
<td>Serum (µl)</td>
<td>3</td>
</tr>
<tr>
<td>Incubation period (s)</td>
<td>17×18</td>
</tr>
<tr>
<td>Reaction type</td>
<td>End point</td>
</tr>
<tr>
<td>Wavelength (nm)</td>
<td>510</td>
</tr>
</tbody>
</table>

Reference range

<table>
<thead>
<tr>
<th>Recommended values</th>
<th>&lt; 200 mg/dL</th>
</tr>
</thead>
<tbody>
<tr>
<td>Upper limit</td>
<td>200-400 mg/dl</td>
</tr>
<tr>
<td>High values</td>
<td>&gt; 400 mg/dl</td>
</tr>
</tbody>
</table>

3.9.4 Determination of serum high density lipoprotein (HDL)

Liquid HDL precipitant for the determination of HDL Cholesterol using Globe diagnostics kit, Italy.
**Intended use**
For the precipitation of LDL and VLDL lipoproteins in serum or plasma with PEG 6000, for subsequent HDL cholesterol determination

**Assay procedure**
1. Pipette into conic test tubes:
   - 0.5 ml serum
   - 0.5 ml precipitating reagent
2. Mix gently by inversion, wait 5 minutes and centrifuge at (3000 rpm) for 20 minutes.
3. Recovery the supernatant for the HDL cholesterol determination as follows:
   - About 0.5 ml of supernatant was transferred to the Mindray BS-120 chemistry autoanalyzer to perform the test according to these parameters:

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Reagent (μl)</td>
<td>200</td>
</tr>
<tr>
<td>supernatant (μl)</td>
<td>3</td>
</tr>
<tr>
<td>Incubation period (s)</td>
<td>17×18</td>
</tr>
<tr>
<td>Reaction type</td>
<td>End point</td>
</tr>
<tr>
<td>Wavelength (nm)</td>
<td>510</td>
</tr>
</tbody>
</table>

**Reference value**
Based on the risk for heart diseases the sequent reference ranges are suggested:

<table>
<thead>
<tr>
<th>Low value (high risk)</th>
<th>Medium value (moderate risk)</th>
<th>High value (low risk)</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 40 mg/dl</td>
<td>40 - 59 mg/dl</td>
<td>&gt; 60 mg/dl</td>
</tr>
</tbody>
</table>
3.9.5 Determination of low density lipoprotein - cholesterol (LDL- c)

Determination of LDL-c was calculated from the primary measurements using the empirical equation:

\[ \text{LDL-c (mg/dl)} = \text{total cholesterol} - \text{triglyceride}/5 - \text{HDL-c} \]

3.10 Hematological analysis

A complete system of reagents of control and calibrator, Cell-Dyn 1700 was used to determine complete blood count (CBC) of children in the laboratories of blood bank.

3.11 Statistical analysis

Data were analyzed using Statistical Package of Social Sciences (SPSS) system (version 18.0). The following statistical tests were applied:

- Frequency distributions
- Independent-samples \( t \)-test
- Chi-square test
- ANOVA test
- Correlation test

The percentage difference was calculated according to the formula:

\[ \text{Percentage difference} = \frac{\text{mean of cases} - \text{mean of controls}}{\text{mean of controls}} \times 100. \]

Probability values (p) were obtained from the student’s table of \( t \) and significance was at \( p < 0.05 \). Range as minimum and maximum values was used.

Microsoft Excel program version 11.0 was used for correlation graphs plotting.
Chapter 4

Results
Chapter 4

Results

4.1 Prevalence of obesity among primary school male children aged 10-12 in West Gaza City

Out of 3409 primary school male children, 148 was found to be obese. Therefore, the prevalence of obesity among primary school male children aged 10-12 in West Gaza City was 4.3%. A total number of 96 obese children parents were respond to questionnaire interview and blood sampling. Therefore the response rate was 64.9%. A total of 96 non obsess children were served as controls. The age of the study population ranged from 10-12 years with mean of 11.1±0.7 for controls and 11.2 ±0.6 for cases (t=0.855, P=0.394).

4.2 Anthropometric measurements of the study population

Table 4.1 shows the anthropometric measurements of the study population. The mean weight (±SD) of cases was 57.0±8.8 kgm compared to 35.7±3.4 kgm of controls (% difference=5.97%, t=22.595 and P=0.000). There was also a significant increase in the mean height of cases compared to controls (1.48±0.07 Vs 1.43±0.06 m, % difference=3.5, t=5.668 and P=0.000). Therefore, BMI of cases was significantly higher than that of controls (25.8±2.7 Vs 17.4±1.0, % difference=48.3, t=29.539 and P=0.000).
Table 4.1 Anthropometric measurements of the study population.

<table>
<thead>
<tr>
<th>Anthropometric measurement</th>
<th>Control (n=96) mean± SD</th>
<th>Case (n=96) mean± SD</th>
<th>% difference</th>
<th>t</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Weight (kg)*</td>
<td>35.7±3.4 (29-44)</td>
<td>57.0±8.8 (43-80)</td>
<td>59.7</td>
<td>22.595</td>
<td>0.000</td>
</tr>
<tr>
<td>Height (m)**</td>
<td>1.43±0.06 (1.28-1.57)</td>
<td>1.48±0.07 (1.30-1.61)</td>
<td>3.5</td>
<td>5.668</td>
<td>0.000</td>
</tr>
<tr>
<td>BMI***</td>
<td>17.4±1.0 (15.0 -20.0)</td>
<td>25.8±2.7 (23.0-34.6)</td>
<td>48.3</td>
<td>29.539</td>
<td>0.000</td>
</tr>
</tbody>
</table>

*Kg: kilogram, ** m: meter. ***BMI: Body mass index: Normal=15.0-20.0, Obese≥23.0 (CDC, 2000). All values are expressed as mean±SD. p<0.05 :significant.

4.3 Sociodemographic data

4.3.1 Education and occupation of the children parents

Education and occupation of the children parents are presented in Table 4.2. Analysis of education status of children fathers showed that 28 (29.2%) fathers of both controls and cases had university degree, and 2 (2.1%) and 1 (1.0%) were illiterate, respectively. For mothers of controls and cases, 16 (16.7) and 18 (18.8) had university degree, and 1 (1.0%) and 0 (0.0%) were illiterate. Parents occupation showed that 68 (70.8%) and 70 (72.9%) fathers of controls and cases were employed compared to unemployed fathers of 28 (29.2%) and 26 (27.1%). The employed mothers of controls and cases were 13 (13.5%) and 12 (12.5%) compared to unemployed of 83 (86.5%) and 84 (87.5%).
Table 4.2 Education and occupation of the children parents of both controls and cases.

<table>
<thead>
<tr>
<th>Sociodemographic aspect</th>
<th>Fathers</th>
<th></th>
<th>Fathers</th>
<th></th>
<th>Mothers</th>
<th></th>
<th>Mothers</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Controls (n=96)</td>
<td>Cases (n=96)</td>
<td>Controls (n=96)</td>
<td>Cases (n=96)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>No. (%)</td>
<td>No. (%)</td>
<td>No. (%)</td>
<td>No. (%)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Education</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Illiterate</td>
<td>2 (2.1)</td>
<td>1 (1.0)</td>
<td>1 (1.0)</td>
<td>0 (0.0)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Primary school</td>
<td>16 (16.7)</td>
<td>12 (12.5)</td>
<td>5 (5.2)</td>
<td>8 (8.3)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Preparatory school</td>
<td>18 (18.8)</td>
<td>26 (27.1)</td>
<td>30 (31.3)</td>
<td>32 (33.3)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Secondary school</td>
<td>32 (33.3)</td>
<td>29 (30.2)</td>
<td>44 (45.8)</td>
<td>38 (39.6)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>University</td>
<td>28 (29.2)</td>
<td>28 (29.2)</td>
<td>16 (16.7)</td>
<td>18 (18.8)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Occupation</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Employed</td>
<td>68 (70.8)</td>
<td>70 (72.9)</td>
<td>13 (13.5)</td>
<td>12 (12.5)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Unemployed</td>
<td>28 (29.2)</td>
<td>26 (27.1)</td>
<td>83 (86.5)</td>
<td>84 (87.5)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

4.3.2 Family income per month and family history of obesity of the study population

Table 4.3 illustrates family income per month and family history of obesity of the study population. Fifty (52.1%), 34 (34.5%) and 12 (12.5%) families of controls had family income <1000, 1000-2000 and >2000 NIS, respectively compared to 42 (43.8%), 38 (39.6%) and 16 (16.7%) families of cases. No significant difference was found between family salaries of controls and cases ($\chi^2=1.489$, $P=0.475$). Concerning family history of obesity, 33 (34.4%) parents of controls reported obesity among their relatives compared to 57 (59.4%) cases who did ($\chi^2=12.047$, $P=0.000$), indicating that family history is a risk factor for obesity.
Table 4.3 Family income /month and family history of obesity of the study population

<table>
<thead>
<tr>
<th>Sociodemographic aspect</th>
<th>Controls (n=96)</th>
<th>Cases (n=96)</th>
<th>$\chi^2$</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No.</td>
<td>%</td>
<td>No.</td>
<td>%</td>
</tr>
<tr>
<td><strong>Family income /month (NIS)</strong>*</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;1000</td>
<td>50</td>
<td>52.1</td>
<td>42</td>
<td>43.8</td>
</tr>
<tr>
<td>1000-2000</td>
<td>34</td>
<td>35.4</td>
<td>38</td>
<td>39.6</td>
</tr>
<tr>
<td>&gt;2000</td>
<td>12</td>
<td>12.5</td>
<td>16</td>
<td>16.7</td>
</tr>
<tr>
<td><strong>Family history of obesity</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>33</td>
<td>34.4</td>
<td>57</td>
<td>59.4</td>
</tr>
<tr>
<td>No</td>
<td>63</td>
<td>65.6</td>
<td>39</td>
<td>40.6</td>
</tr>
</tbody>
</table>

*NIS: New Israeli Shekels, P>0.05: not significant, P<0.05: significant.

4.4 Feeding and feeding habits of study population

4.4.1 Breast feeding and family health education about obesity

Table 4.4 illustrates breast feeding and family health education about obesity of the study population. Seventy nine (81.3%), 12 (13.5%) and 5 (5.2%) controls had exclusive breast feeding, non-exclusive breast feeding and artificial milk, respectively compared to 69 (72.9%), 20 (19.8%) and 7 (7.3%) cases. No significant difference was found between controls and cases in terms of breast feeding ($\chi^2=3.009$, P=0.222). Family health education on obesity showed that 11 (11.5%) families of controls had health education versus to 15 (15.6%) cases ($\chi^2=0.712$, P=0.399).
Table 4.4 Breast feeding and family health education about obesity

<table>
<thead>
<tr>
<th>Breast feeding and health education</th>
<th>Controls (n=96)</th>
<th>Cases (n=96)</th>
<th>(\chi^2)</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No.</td>
<td>%</td>
<td>No.</td>
<td>%</td>
</tr>
<tr>
<td><strong>Breast feeding</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Exclusive breast feeding*</td>
<td>79</td>
<td>81.3</td>
<td>69</td>
<td>72.9</td>
</tr>
<tr>
<td>Non-exclusive**</td>
<td>12</td>
<td>13.5</td>
<td>20</td>
<td>19.8</td>
</tr>
<tr>
<td>Artificial milk</td>
<td>5</td>
<td>5.2</td>
<td>7</td>
<td>7.3</td>
</tr>
<tr>
<td><strong>Family health education</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>11</td>
<td>11.5</td>
<td>15</td>
<td>15.6</td>
</tr>
<tr>
<td>No</td>
<td>85</td>
<td>88.5</td>
<td>81</td>
<td>84.4</td>
</tr>
</tbody>
</table>

*Exclusive breastfeeding: only breast milk, no other liquid or solid from any other source enters the child’s mouth (Labbok, 2000).

**Non-exclusive breastfeeding: children received drinks/foods with breast milk.

P>0.05: not significant

4.4.2 Drinking and eating among the study population

Drinking and eating among the study population are summarized in Table 4.5. A total of 78 (81.3%) controls drank soft drink compared to 80 (83.3%) cases \(\chi^2=0.143, \ P=0.705\). When parents asked about the frequency of soft drink, 1 (1.3%), 19 (24.4%) and 58 (74.3%) controls drink daily, 2-3 times/week and once/week compare to their counterparts of 7 (8.8%), 21 (26.3%) and 52 (65.0%), respectively \(\chi^2=3.378, \ P=0.185\). Ninety one (94.8%) controls ate sweets versus 93 (96.9%) cases \(\chi^2=0.130, \ P=0.718\). Thirty one (34.1%), 41(45.1%) and 19 (20.9%) controls ate sweets daily, 2-3 times/week and once/week versus their counterparts of 42 (45.2%), 30 (32.3%) and 21 (22.6%), respectively \(\chi^2=3.440, \ P=0.179\). In addition, 90 (93.8%) controls ate indomy compared to 87 (90.6%) cases \(\chi^2=0.651, \ P=0.420\). Three (3.3%), 19 (21.1%) and 68 (75.6%) controls ate indomy daily, 2-3 times/week and once/week compared to their counterparts of 6 (6.9%), 30 (34.9%) and 51 (58.6%), respectively \(\chi^2=4.553, \ P=0.103\).
### Table 4.5 Drinking and eating among the study population

<table>
<thead>
<tr>
<th>Feeding items</th>
<th>Controls (n=96)</th>
<th>Cases (n=96)</th>
<th>χ²</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No.</td>
<td>%</td>
<td>No.</td>
<td>%</td>
</tr>
<tr>
<td><strong>Drinking soft drink</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>78</td>
<td>81.3</td>
<td>80</td>
<td>83.3</td>
</tr>
<tr>
<td>No</td>
<td>18</td>
<td>18.8</td>
<td>16</td>
<td>16.7</td>
</tr>
<tr>
<td><strong>If yes</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Daily</td>
<td>1</td>
<td>1.3</td>
<td>7</td>
<td>8.8</td>
</tr>
<tr>
<td>2-3 times/week</td>
<td>19</td>
<td>24.4</td>
<td>21</td>
<td>26.3</td>
</tr>
<tr>
<td>Once/week</td>
<td>58</td>
<td>74.3</td>
<td>52</td>
<td>65.0</td>
</tr>
<tr>
<td><strong>Eating sweets</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>91</td>
<td>94.8</td>
<td>93</td>
<td>96.9</td>
</tr>
<tr>
<td>No</td>
<td>5</td>
<td>5.2</td>
<td>3</td>
<td>3.1</td>
</tr>
<tr>
<td><strong>If yes</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Daily</td>
<td>31</td>
<td>34.1</td>
<td>42</td>
<td>45.2</td>
</tr>
<tr>
<td>2-3 times/week</td>
<td>41</td>
<td>45.1</td>
<td>30</td>
<td>32.3</td>
</tr>
<tr>
<td>Once/week</td>
<td>19</td>
<td>20.9</td>
<td>21</td>
<td>22.6</td>
</tr>
<tr>
<td><strong>Eating indomy</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>90</td>
<td>93.8</td>
<td>87</td>
<td>90.6</td>
</tr>
<tr>
<td>No</td>
<td>6</td>
<td>6.3</td>
<td>9</td>
<td>9.4</td>
</tr>
<tr>
<td><strong>If yes</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Daily</td>
<td>3</td>
<td>3.3</td>
<td>6</td>
<td>6.9</td>
</tr>
<tr>
<td>2-3 times/week</td>
<td>19</td>
<td>21.1</td>
<td>30</td>
<td>34.9</td>
</tr>
<tr>
<td>Once/week</td>
<td>68</td>
<td>75.6</td>
<td>51</td>
<td>58.6</td>
</tr>
</tbody>
</table>

P>0.05:not significant.
4.4.3 Feeding habits of the study population

Table 4.6 provides feeding habits of the study population. A total of 87 (90.6%) and 9 (9.4%) controls ate 2-3 meals/day and > 3 meals/day, respectively compared to 73 (76.0%) and 23 (24.0%) cases. The decrease between controls and cases was statistically significant with $\chi^2=7.350$ and $P=0.007$. Sixty eight (70.8%) and 52 (54.2%) controls ate school meals and eating while watching television, respectively compared to their counterparts of 64 (66.7%) and 65 (67.7%) with $\chi^2=0.388$, $P=0.533$ and $\chi^2=3.698$, $P=0.054$, respectively.

<table>
<thead>
<tr>
<th>Feeding habits</th>
<th>Controls (n=96)</th>
<th>Cases (n=96)</th>
<th>$\chi^2$</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No.</td>
<td>%</td>
<td>No.</td>
<td>%</td>
</tr>
<tr>
<td>meals/day</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2-3</td>
<td>87</td>
<td>90.6</td>
<td>73</td>
<td>76.0</td>
</tr>
<tr>
<td>&gt;3</td>
<td>9</td>
<td>9.4</td>
<td>23</td>
<td>24.0</td>
</tr>
<tr>
<td>Eating school meal</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>68</td>
<td>70.8</td>
<td>64</td>
<td>66.7</td>
</tr>
<tr>
<td>No</td>
<td>28</td>
<td>29.2</td>
<td>32</td>
<td>33.3</td>
</tr>
<tr>
<td>Eating while watching television</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>52</td>
<td>54.2</td>
<td>65</td>
<td>67.7</td>
</tr>
<tr>
<td>No</td>
<td>44</td>
<td>45.8</td>
<td>31</td>
<td>32.3</td>
</tr>
</tbody>
</table>

$P>0.05$: not significant, $P<0.05$: significant

4.5 Physical activity among the study population

Physical activity among the study population is shown in Table 4.7. Seventy three (76.0%) and 68 (70.8%) controls doing exercise and playing football, respectively versus cases of 33 (34.4%) and 44 (45.8%). The differences
between controls and cases were significant ($\chi^2=0.33.699$, $P=.0000$ and $\chi^2=12.343$, $P=0.000$, respectively).

Table 4.7 Physical activity among the study population

<table>
<thead>
<tr>
<th>Physical activity</th>
<th>Controls (n=96)</th>
<th>Cases (n=96)</th>
<th>$\chi^2$</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No.</td>
<td>%</td>
<td>No.</td>
<td>%</td>
</tr>
<tr>
<td><strong>Doing exercise</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>73</td>
<td>76.0</td>
<td>33</td>
<td>34.4</td>
</tr>
<tr>
<td>No</td>
<td>23</td>
<td>24.0</td>
<td>63</td>
<td>65.6</td>
</tr>
<tr>
<td><strong>Playing football</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>68</td>
<td>70.8</td>
<td>44</td>
<td>45.8</td>
</tr>
<tr>
<td>No</td>
<td>28</td>
<td>29.2</td>
<td>52</td>
<td>54.2</td>
</tr>
</tbody>
</table>

, $P<0.05$: significant.

4.6 Leptin and lipid profile of the Study population

Serum leptin and lipid profile including, total cholesterol, triglycerides, low density lipoproteins (LDL) and high density lipoproteins (HDL) are listed in Table 4.8. The mean level of leptin was significantly elevated in cases compared to controls (31.5±16.3 Vs 6.9±6.1 ng/ml, % difference=356.5%, and $P=0.000$). Similarly, the mean levels of total cholesterol, triglycerides and LDL were significantly increased in cases compared to controls (153.2±34.4 Vs 138.2±22.5 mg/dl, % difference=10.9% and $P=0.000$; 117.7±34.9 Vs 100.3±27.4 mg/dl, % difference=17.3% and $P=0.000$; and 84.9±35.9 Vs 69.6±21.1 mg/dl, % difference=22.0 and $P=0.000$, respectively). In contrast, the mean level of HDL was significantly decreased in cases compared to controls (44.2±13.5 Vs 49.5±12.8 mg/dl, % difference=10.7 and $P=0.000$).
Table 4.8 Leptin and lipid profile of the Study population

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Control (n=96) mean±SD</th>
<th>Case (n=96) mean±SD</th>
<th>% difference</th>
<th>T</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Leptin (ng/ml)</td>
<td>6.9±6.1 (0.9-32.1)</td>
<td>31.5±16.3 (1.3-49.4)</td>
<td>356.5</td>
<td>13.479</td>
<td>0.000</td>
</tr>
<tr>
<td>Cholesterol (mg/dl)</td>
<td>138.2±22.5 (100-195)</td>
<td>153.2±34.4 (92-249)</td>
<td>10.9</td>
<td>3.628</td>
<td>0.000</td>
</tr>
<tr>
<td>Triglyceride (mg/dl)</td>
<td>100.3±27.4 (39-197)</td>
<td>117.7±34.9 (65-235)</td>
<td>17.3</td>
<td>3.925</td>
<td>0.000</td>
</tr>
<tr>
<td>LDL (mg/dl) *</td>
<td>69.6±21.1 (34-123)</td>
<td>84.9±35.9 (27-173)</td>
<td>22.0</td>
<td>3.598</td>
<td>0.000</td>
</tr>
<tr>
<td>HDL (mg/dl) **</td>
<td>49.5±12.8 (31-90)</td>
<td>44.2±13.5 (23-88)</td>
<td>-10.7</td>
<td>-2.826</td>
<td>0.005</td>
</tr>
</tbody>
</table>

*LDL: Low density lipoprotein, **HDL: High density lipoprotein. All values are expressed as mean ±SD. P<0.05: significant.

4.7 White blood cells and blood platelets of the Study population

Table 4.9 demonstrates white blood cells count and blood platelets in cases and controls. White blood cell count showed non significant increase in cases compared to controls (7.2±2.0 Vs 6.9±2.0 X10^3 cell/µl, % difference=4.3, t=1.328 and p=0.186). Like white blood cells, blood platelets were increased in the cases compared to the controls (356.2±82.6 and 326.5±85.3 % difference=9.1). However, this increase was significant (t=2.490 and P=0.014).
Table 4.9 White blood cells and blood platelets of the Study population

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Control (n=96) mean±SD</th>
<th>Case (n=96) mean±SD</th>
<th>% difference</th>
<th>t</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>WBC(×10³cell/µl)</td>
<td>6.9±2.0 (2.3-11.8)</td>
<td>7.2±2.0 (3.5-12.7)</td>
<td>4.3</td>
<td>1.328</td>
<td>0.186</td>
</tr>
<tr>
<td>PLT(×10³cell/µl)</td>
<td>326.5±85.3 (108-602)</td>
<td>356.2±82.6 (159-726)</td>
<td>9.1</td>
<td>2.490</td>
<td>0.014</td>
</tr>
</tbody>
</table>

WBC=white blood cell (×10⁶ cell/µl); PLT=platelet (×10³ cell/µl). P<0.05: significant, p>0.05 not significant. All values are expressed as mean ±SD.

4.8 Primary and secondary blood indices of the Study population

Primary and secondary blood indices of the cases and the controls are demonstrated in Table 4.10. For the primary blood indices, the mean of red blood cell count was significantly increased in cases compared to controls (4.87±0.44 Vs 4.75±0.42 ×10⁶cell/µl, % difference=2.5, t=1.996 and p=0.047). There were no significant changes in hemoglobin content and hematocrit in cases compared to controls, recording % differences of -1.7 and -0.8, respectively. (11.76±0.78 Vs 11.96±0.82 g/dl, t =-1.707, P= 0.089 and 36.9±2.6 Vs 37.2±2.9%, t =-0.961, P= 0.490, respectively). Secondary blood indices including MCV, MCH and MCHC were also found to be lower in cases compared to controls registering % differences of 3.2, 4.0 and 0.9, respectively (75.9±5.2, 24.3±2.3, 31.9±1.4 Vs 78.4±4.1 fl, 25.3±1.9 pg, 32.2±1.4 g/dl). Such changes were significant for MCV and MCH not significant for MCHC (t=3.671, p=0.000; t=3.466, p=0.001 and t= 1.612, P=0.109, respectively).
Table 4.10. Primary and secondary blood indices of the study population

<table>
<thead>
<tr>
<th>Blood parameter</th>
<th>Control mean±SD</th>
<th>Case mean±SD</th>
<th>% Difference</th>
<th>t</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>RBCs count (X10⁶cell/µl)</td>
<td>4.75±0.42 (2.72-5.71)</td>
<td>4.87±0.44 (3.77-6.34)</td>
<td>2.5</td>
<td>1.996</td>
<td>0.047</td>
</tr>
<tr>
<td>Range (min-max)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>HGB content (g/dl)</td>
<td>11.96±0.82 (9.6-13.7)</td>
<td>11.76±0.78 (9.3-13.9)</td>
<td>-1.7</td>
<td>1.707</td>
<td>0.089</td>
</tr>
<tr>
<td>Range (min-max)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hct (%)</td>
<td>37.2±2.9 (21.0-42.4)</td>
<td>36.9±2.6 (30.4-43.5)</td>
<td>-0.8</td>
<td>0.961</td>
<td>0.490</td>
</tr>
<tr>
<td>Range (min-max)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>MCV (fl)</td>
<td>78.4±4.1 (65.9-87.6)</td>
<td>75.9±5.2 (56.2-87.2)</td>
<td>-3.2</td>
<td>3.671</td>
<td>0.000</td>
</tr>
<tr>
<td>Range (min-max)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>MCH (pg)</td>
<td>25.3±1.9 (19.7-30.2)</td>
<td>24.3±2.3 (17.2-29.6)</td>
<td>-4.0</td>
<td>3.466</td>
<td>0.001</td>
</tr>
<tr>
<td>Range (min-max)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>MCHC (g/dl)</td>
<td>32.2±1.4 (30.0-37.1)</td>
<td>31.9±1.4 (29.6-38.0)</td>
<td>-0.9</td>
<td>1.612</td>
<td>0.109</td>
</tr>
<tr>
<td>Range (min-max)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

RBC=Red blood cell; HGB=hemoglobin; Hct=Heamtocrit; MCV=mean corpuscular volume; MCH=mean corpuscular hemoglobin; MCHC=mean corpuscular hemoglobin concentration. P<0.05: significant, p>0.05 not significant. All values are expressed as ±

4.9 Socioeconomic parameters of obese children in relation to their body mass index

Table 4.11 provides socioeconomic parameters of obese children in relation to their body mass index. It is noted that the mean of BMI was the highest in children who their mothers had primary school education (26.5±3.3 kg/m²) and also in children of families had salary <1000 NIS (26.2±2.6 kg/m²). However, the relation of BMI with these socioeconomic parameters as well as with occupation and family history of obesity was not significant (P>0.05).
Table 4.11 Socioeconomic parameters of parents obese children in relation to their body mass index

<table>
<thead>
<tr>
<th>Socioeconomic parameters</th>
<th>Cases (n=96)</th>
<th>BMI (mean±SD)</th>
<th>Test</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Education level of the parents</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Primary school</td>
<td>08</td>
<td>26.5±3.3</td>
<td>F</td>
<td>0.191</td>
</tr>
<tr>
<td>Preparatory school</td>
<td>32</td>
<td>25.8±2.6</td>
<td></td>
<td>0.902</td>
</tr>
<tr>
<td>Secondary school</td>
<td>38</td>
<td>25.7±2.8</td>
<td></td>
<td></td>
</tr>
<tr>
<td>University</td>
<td>18</td>
<td>25.8±2.6</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Occupation of the parents</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Employed</td>
<td>12</td>
<td>25.2±2.9</td>
<td>t</td>
<td>0.824</td>
</tr>
<tr>
<td>Unemployed</td>
<td>84</td>
<td>25.9±2.7</td>
<td></td>
<td>0.412</td>
</tr>
<tr>
<td><strong>Family salary/month (NIS)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;1000</td>
<td>42</td>
<td>26.2±2.6</td>
<td>F</td>
<td>0.753</td>
</tr>
<tr>
<td>1000-2000</td>
<td>38</td>
<td>25.7±3.0</td>
<td></td>
<td>0.474</td>
</tr>
<tr>
<td>&gt;2000</td>
<td>16</td>
<td>25.3±2.2</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Family history of obesity</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>57</td>
<td>25.9±2.5</td>
<td>t</td>
<td>0.358</td>
</tr>
<tr>
<td>No</td>
<td>39</td>
<td>25.7±3.1</td>
<td></td>
<td>0.721</td>
</tr>
</tbody>
</table>

*P>0.05:not significant

4.10 Feeding and feeding habits of obese children in relation to their body mass index

4.10.1 Breast feeding and health education of obese children in relation to their BMI

Table 4.12 shows breast feeding and health education of obese children in relation to their body mass index. The mean of BMI was the lowest in the children who had exclusive breast feeding (25.3±2.2), followed by that of children who had non-exclusive breast feeding (23.5±2.9) and then by artificial milk (26.0±2.7). However, group difference was not significant (F=0.377, P=
0.687). Like breast feeding, the difference in the mean BMI regarding family health education was not significant with higher BMI in children who their families did not have health education (26.6±3.5 Vs 25.7±2.5, t=1.249, P=0.215).

Table 4.12 Breast feeding and health education of obese children in relation to their body mass index

<table>
<thead>
<tr>
<th>Breast feeding and health education</th>
<th>Cases (n=96)</th>
<th>test</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No.</td>
<td>BMI (mean±SD)</td>
<td></td>
</tr>
<tr>
<td>Breast feeding</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Exclusive breast feeding</td>
<td>69</td>
<td>25.3±2.2</td>
<td>F</td>
</tr>
<tr>
<td>Non-exclusive</td>
<td>20</td>
<td>25.5±2.9</td>
<td></td>
</tr>
<tr>
<td>Artificial milk</td>
<td>7</td>
<td>26.0±2.7</td>
<td></td>
</tr>
<tr>
<td>Family health education</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>15</td>
<td>25.7±2.5</td>
<td>t</td>
</tr>
<tr>
<td>No</td>
<td>81</td>
<td>26.6±3.5</td>
<td></td>
</tr>
</tbody>
</table>

P>0.05:not significant

4.10.2 Drinking and eating of obese children in relation to their body mass index

Drinking and eating among of obese children in relation to their body mass index are presented in Table 4.13. The means of BMI of obese children who drunk soft drink, ate sweets, and ate chips and indomy were higher than those who did not (26.0±2.7, 27.7±2.6, 25.8±2.7 Vs 25.0±2.6, 25.8±2.7 and 25.6±2.4 kg/m², respectively). However, the differences between groups were not significant (t=1.372, P=0.173; t=1.242, P=0.218 and t=0.317, P=0.317, respectively).
Table 4.13 Drinking and eating among obese children in relation to their body mass index

<table>
<thead>
<tr>
<th>Feeding habit</th>
<th>Cases (n=96)</th>
<th>BMI (mean±SD)</th>
<th>t</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Drinking soft drink</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>80</td>
<td>26.0±2.7</td>
<td>1.372</td>
<td>0.173</td>
</tr>
<tr>
<td>No</td>
<td>16</td>
<td>25.0±2.6</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Eating sweets</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>93</td>
<td>27.7±2.6</td>
<td>1.242</td>
<td>0.218</td>
</tr>
<tr>
<td>No</td>
<td>3</td>
<td>25.8±2.7</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Eating chips and indomy</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>87</td>
<td>25.8±2.7</td>
<td>0.317</td>
<td>0.317</td>
</tr>
<tr>
<td>No</td>
<td>9</td>
<td>25.6±2.4</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

P>0.05: not significant

4.10.3 Feeding habits of obese children in relation to their body mass index

Table 4.14 showed feeding habits of obese children in relation to their body mass index. The mean of BMI of obese children who ate while watching television was 26.4±2.8 compared to those who did not (24.7±2.2). The difference between the two groups was significant (t=2.962, P=0.004). On the other hand, the interactions of BMI with the number of meals/day and eating school meal were not significant (25.7±2.8 Vs 26.1±2.6, t=0.615, P=0.540 and 25.7±2.4 Vs 26.1±3.2, t=0.726, P=0.470, respectively).
Table 4.14 Feeding habits of obese children (cases) in relation to their body mass index

<table>
<thead>
<tr>
<th>Feeding habits</th>
<th>Cases (n=96)</th>
<th>t</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No.</td>
<td>BMI (mean±SD)</td>
<td></td>
</tr>
<tr>
<td>meals/day</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2-3</td>
<td>73</td>
<td>25.7±2.8</td>
<td>0.615</td>
</tr>
<tr>
<td>&gt;3</td>
<td>23</td>
<td>26.1±2.6</td>
<td></td>
</tr>
<tr>
<td>Eating school meal</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>64</td>
<td>25.7±2.4</td>
<td>0.726</td>
</tr>
<tr>
<td>No</td>
<td>32</td>
<td>26.1±3.2</td>
<td></td>
</tr>
<tr>
<td>Eating while watching television</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>65</td>
<td>26.4±2.8</td>
<td>2.962</td>
</tr>
<tr>
<td>No</td>
<td>31</td>
<td>24.7±2.2</td>
<td></td>
</tr>
</tbody>
</table>

P>0.05: not significant, P<0.05: significant

4.11 Physical activity of obese children (cases) in relation to their body mass index

Physical activity of obese children in relation to their body mass index is pointed out in Table 4.15. The interaction of BMI with physical activity of obese children was not significant, with higher BMI in children who did not exercise and play football (25.8±2.7 Vs 25.6±2.8, t=0.027, P=0.979 and 26.1±2.8 Vs 25.5±2.6 t=1.100, P=0.274, respectively).
Table 4.15 Physical activity of obese children (cases) in relation to their body mass index

<table>
<thead>
<tr>
<th>Physical activity</th>
<th>Cases (n=96)</th>
<th>t</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No.</td>
<td>BMI (mean±SD)</td>
<td></td>
</tr>
<tr>
<td>Doing exercise</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>33</td>
<td>25.6±2.8</td>
<td>0.027</td>
</tr>
<tr>
<td>No</td>
<td>63</td>
<td>25.8±2.7</td>
<td></td>
</tr>
<tr>
<td>Playing football</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>44</td>
<td>25.5±2.6</td>
<td>1.100</td>
</tr>
<tr>
<td>No</td>
<td>52</td>
<td>26.1±2.8</td>
<td></td>
</tr>
</tbody>
</table>

P>0.05: significant

4.12 The correlation of BMI with leptin hormone and lipid profile of obese children (cases, n=96).

The correlations of BMI with leptin hormone and lipid profile including total cholesterol, triglyceride, high density lipoprotein (HDL) and low density lipoprotein (LDL) of obese children are presented in Table 4.16. There was a positive significant correlation between BMI and levels of leptin hormone with r=0.305 and P=0.003 (Figure 4.1). On the other hand, weak not significant correlations were found between BMI and total cholesterol, triglyceride, HDL and LDL (r=0.111, P=0.275; r=0.055, P=0.592; r=-0.063, P=0.538 and r=0.131, P=0.200, respectively).
Table 4.16. The correlation of BMI with leptin hormone and lipid profile of obese children (cases, n=96).

<table>
<thead>
<tr>
<th>Parameter</th>
<th>R</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Leptin (ng/ml)</td>
<td>0.305</td>
<td>0.003</td>
</tr>
<tr>
<td>Total cholesterol (mg/dl)</td>
<td>0.111</td>
<td>0.275</td>
</tr>
<tr>
<td>Triglyceride (mg/dl)</td>
<td>0.055</td>
<td>0.592</td>
</tr>
<tr>
<td>HDL (mg/dl)</td>
<td>-0.063</td>
<td>0.538</td>
</tr>
<tr>
<td>LDL (mg/dl)</td>
<td>0.131</td>
<td>0.200</td>
</tr>
</tbody>
</table>

P>0.05: not significant, P<0.05: significant

Figure 4.1 Correlation between BMI and leptin of obese children

(r=0.305, P=0.003)
4.13 The correlation of leptin hormone with lipid profile of obese children

The correlations of leptin with lipid profile including total cholesterol, triglyceride, high density lipoprotein HDL and low density lipoprotein LDL of obese children are illustrated in Table 4.17. There was a positive significant correlation between leptin and total cholesterol with \( r=0.202 \) and \( P=0.049 \) (Figure 4.2). On the other hand, weak not significant correlations were found between leptin and triglyceride, HDL and LDL (\( r=0.081, P=0.437 \); \( r=-0.089, P=0.389 \) and \( r=0.189, P=0.067 \), respectively).

Table 4.17. The correlation of leptin hormone with lipid profile of obese children (cases, \( n=96 \)).

<table>
<thead>
<tr>
<th>Parameter</th>
<th>R</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total cholesterol (mg/dl)</td>
<td>0.202</td>
<td>0.049</td>
</tr>
<tr>
<td>Triglyceride (mg/dl)</td>
<td>0.081</td>
<td>0.437</td>
</tr>
<tr>
<td>HDL (mg/dl)</td>
<td>-0.089</td>
<td>0.389</td>
</tr>
<tr>
<td>LDL (mg/dl)</td>
<td>0.189</td>
<td>0.067</td>
</tr>
</tbody>
</table>

\( P>0.05: \) not significant, \( P<0.05: \) significant
Figure 4.2 Correlation between leptin hormone with total cholesterol of obese children

(r=0.202, P=0.049)
Chapter 5

Discussion
Chapter 5

Discussion

Obesity is a major public health problem resulting in serious social, physical and psychological damages. The prevalence of obesity and overweight among adult and children is increasing in developed and developing countries including Gaza strip. Despite that, there are under-diagnosis and under-reporting of the disease. Data on obesity were limited to annual reports emerged from the Palestinian Ministry of Health. Recently, few studies have been carried out on adult obesity in the Gaza Strip (Zabut et al., 2007; Zabut et al., 2009 and Al-Jedi, 2010). However, childhood obesity was not studied particularly at the age group 10-12 years. Therefore, to our knowledge this is the first study to assess obesity among primary school male children aged 10-12 years in West Gaza City.

The present study is a case control investigation included 96 obese children (BMI $\geq 23.0$ kg/m$^2$) and 96 control (non obese individuals, BMI=15.0-20.0 kg/m$^2$) matched with age. Body mass index of children differs from adult, the assessment of weight status is much more complex. This is because children are growing and the growth patterns (and hence the BMI) of children differs by age (Parliamentary Office of Science and Technology, 2003 and Barbara and Clyde Park., 2009). The pattern of growth is dependent upon the sex of the child since the growth pattern for boys is very different from the growth pattern for girls. Therefore, BMI for male children in present study was calculated by a computer graphs program specially design to this purpose (National Health Center for Statistics in collaboration with the National Centre for Chronic Disease Prevention and Health Promotion, 2000).

A very low level of illiteracy was found among parents of both cases and controls, reflecting a well educated community. According to the Palestinian Central Bureau of Statistics (PCBS), in 1997, approximately
over 90% of Gaza's population over the age of 10 was literate. Of the city's population, 140,848 were enrolled in schools (39.8% in elementary school, 33.8% in secondary school, and 26.4% in high school). About 11,134 people received bachelor diplomas or higher diplomas. About one third of children fathers and more than three quarters of children mothers were unemployed, and around half of families had monthly income less than 1000 NIS, reflecting poverty and unemployment crisis in Gaza Strip. Giacaman et al., (2009) reported that unemployment in Gaza Strip was around 33% of the active workforce in 2007, and rose to 37% in 2008. Such unemployment crisis is expected to exceed 50% in 2011 as a result of the continuous siege on economic conditions in Gaza Strip. They added that 52% of families (40% in the West Bank and 74% in the Gaza Strip) were living below the poverty line of US$ 3·15 per person per day.

Regarding family history of obesity, the number of cases reported obesity among their relatives was significantly higher than controls, indicating that family history could be a risk factor of obesity. These findings are in agreement with that reported by Kumar et al. (2010); Zabut et al. (2009) and Al-Jedi (2010). Although the number of cases who exclusively breastfed was lower than controls, the difference was not significant. This finding is inconsistent with Arenze et al. (2004) who concluded that breast-feeding seems to have a small but consistent protective effect against obesity in children.

Feeding and feeding habits of the study population revealed higher number of cases who drunk soft drink and ate sweets than their counterparts with no significant differences. On the other hand the number of cases who had more than 3 meals/day was significantly higher than controls. Eating while watching television showed a borderline significant difference with higher number of cases. Gillis and Bar-Or (2003) reported that obese children and adolescents consumed significantly more servings of meat and alternatives, grain products, food away from home, sugar-sweetened drinks and potato chips which contributed to a higher calorie, fat and sugar intake compared to non-obese children and adolescents. In addition, Dennison et
al. (2002) emphasized that a television in the child’s bedroom is a strong marker of increased risk of being overweight. This view supports the result presented in our study that eating while watching television was significantly associated with BMI in obese children. Three potential mechanisms have been suggested to link television viewing and obesity. The first mechanism is reduced energy expenditure from television viewing displacing physical activity (Epstein et al., 1997). The second mechanism is increasing dietary energy intake from eating during viewing or from the effect of food advertising. A third potential mechanism is that television viewing decreases resting metabolic rate (Buchowski and Sun, 1996). However, the relation between BMI and other items of feeding and feeding habits was not significant.

Concerning physical activity in terms of doing exercise and playing football, the present results revealed that the number of cases doing physical activity was significantly lower than controls. This indicates that physical activity has a preventive effect on obesity. However, this effect was masked within obese children. Such inverse relationship between physical activity and obesity was exhibited by many authors (Trost et al., 2001; Lioret et al., 2007 and Fernandes and Sturm, 2011). In the context of etiology of obesity, Afridi and Khan, (2004) reported that obesity develops as a result of a complex interaction between a person’s genes and the environment characterized by long-term energy imbalance due to excessive caloric consumption, insufficient energy output (sedentary lifestyle, low resting metabolic rate) or both. In addition, Yu et al. (2002) pointed particularly to the potential benefit of increasing physical exercise time relative to sedentary activities to reduce the prevalence of childhood obesity.

The present data showed that the mean level of leptin was markedly elevated in cases compared to controls. This finding is agreement with other studies (Falorni et al., 1997; Dubey et al., 2007 and Yamborisut et al., 2009). Leptin was considered as a potential biomarker for childhood obesity (Venner et al., 2006). In obese subjects serum levels of the adipocyte hormone leptin are increased in proportion to body fat stores as a result of increased production in enlarged fat cells from obese subjects (Fried et al.,
Several studies indicated that insulin and glucocorticoids work directly on adipose tissue to upregulate in a synergistic manner leptin mRNA levels and rates of leptin secretion in human adipose tissue over the long term. Thus, the increased leptin expression observed in obesity could result from the chronic hyperinsulinemia and increased cortisol turnover (Tan et al., 1999 and Rance et al., 2007).

When related to BMI, leptin showed positive correlation with BMI in obese children. This supports the previous view that leptin is increased in proportion to body fat stores as a result of increased production in enlarged fat cells from obese subjects. Similar results were documented by Xua et al (2004) and Dubey et al. (2007). In addition, Pilcova et al. (2003) reported that leptin levels correlated positively with the body mass index before and after weight reduction. Serum leptin also shows a strong relationship to fat distribution in boys and in girls.

The mean levels of total cholesterol, triglycerides and LDL were significantly increased in cases compared to controls whereas the mean level of HDL was significantly decreased in cases. These findings are in accordance with that adressed by Bhatti et al. (2001); Shilian et al. (2004) and Dubey et al. (2007). The lipid disturbance observed in the current study could be attributed to the development of insulin resistant in obese children. Garcés et al. (2005) found that insulin resistance was significantly higher in obese children. Hyperinsulinemia is known to enhance hepatic very-low-density lipoprotein synthesis and thus may directly contribute to the increased plasma triglyceride and LDL cholesterol levels (Stalder et al., 1981). Resistance to the action of insulin on lipoprotein lipase in peripheral tissues may also contribute to elevated triglyceride and LDL cholesterol levels (Sadur et al., 1984).

Data presented here showed that WBC and blood platelets increased in obese children compared to controls. However, the increase in blood platelets was significant. Red blood cells count were significantly increased whereas MCV and MCH were significantly decreased in obese children.
Wilson et al. (1997) found positive correlation between WBC count with percentage of body fat. In addition, Herishanu et al. (2006) recognized obesity as a possible cause for reactive leukocytosis. Increased blood platelets count was also suggested to be significantly associated with obesity (Luenda et al., 2007). The changes observed in the primary and secondary blood indices may be related to the recorded increase in leptin levels in obese children which may stimulate the proliferation of haemopoietic stem cells (Montoye et al., 2006).
Chapter 6

Conclusions and Recommendations
Chapter 6

Conclusions and Recommendations

6.1 Conclusions

* The prevalence of obesity among primary school male children aged 10-12 years in West Gaza City was 4.3%.

* Body mass index of obese children (cases) was 25.8±2.7 whereas that of control children was 17.4±1.0.

* Family history was a risk factor of obesity.

* The number of cases who exclusively breastfed was lower than controls whereas those who had more than 3 meals/day was significantly higher than controls, implying that lack of breast feeding and having ≥3 meals/day could contribute to obesity.

* Eating while watching television showed a borderline significant difference with higher number of cases. However, eating while watching television was significantly associated with BMI in obese children.

* Concerning physical activity in terms of doing exercise and playing football, the number of cases was significantly lower than controls, indicating that physical activity has a preventive effect on obesity.

* The mean level of leptin was markedly elevated in cases compared to controls. When related to BMI, leptin showed positive correlation with BMI and total cholesterol in obese children.

* The mean levels of total cholesterol, triglycerides and LDL were significantly increased in cases compared to controls whereas the mean level of HDL was significantly decreased in cases.
* White blood cells and blood platelets counts were increased in obese children compared to controls. However, the increase in blood platelets was significant. Red blood cells were significantly increased whereas MCV and MCH were significantly decreased in obese children.
6.2 Recommendations

* Launching of health education programs among children parents on childhood obesity in terms of avoiding factor contributing to weight gain and the benefit of follow up a healthy diet.

* Eating >3 meals/day as well as eating while watching television must be avoidable as can as possible.

* Exclusive breast feeding is appreciated at least for the first six months.

* Regular physical activity is highly recommend.
Chapter 7

References
Chapter 7

References


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72- Rance KA., Johnstone AM., Murison S., Duncan JS., Wood SG., and Speakman JR. (2007): Plasma leptin levels are related to body


Annex 1

This questionnaire was designed to Study obesity and factors that help in its prevalence among the students of primary schools in the west of Gaza City.

Students parent / I will very grateful if you help me in completing this study (Master research heath science / the Islamic University) Which specializes in the prevalence of obesity among primary school students in the Gaza Strip, its goal in to identify the factors affecting the prevalence of obesity among the children, I will be very grateful if you help your children filling the questionnaire and taking blood sample from them to do some biochemical analysis.

Note: Please read carefully the paragraphs before answering put an X in the box when the desired answers selected.

The student's name: -------------------------- Address: ------------------

School Name: ----------------------------- Grade: ---------------------

Age: ---------------------------

1. Father's education: ☐ university ☐ preparatory ☐ secondary ☐ primary ☐ uneducated

2. Mother's education: ☐ university ☐ preparatory ☐ secondary ☐ primary ☐ uneducated

3. Father job: ☐ Government officer ☐ special work ☐ Factor ☐ Retired

4. Mother job: ☐ Government officer ☐ special work ☐ Factor ☐ Retired

5. The family income: ☐ less than 1000 shekels ☐ From 1000 1500 shekels ☐ From 1500 2000 shekels ☐ From 2000 shekels and over

6. Is there anyone in the family suffering from obesity? ☐ Yes ☐ No

If the answer is yes..................... What is degree of relationship?:

☐ father / mother ☐ brother / sister ☐ grandfather / grandmother ☐ uncle / aunt-free / aunt
7. Do you drink soft drinks? □ Yes □ No

If the answer is yes-----------------------How many time?

□ Once a week □ 2-4 times per week □ daily

8. Do you have sweet snacks (biscuits, cake, chocolate)? □ Yes □ No

If the answer is yes-----------------------How many time?

□ Once a week □ 2-4 times per week □ daily

9. How many packets chips and andome in do you have today? □ 1 □ 2-3 □ more than 3

10. Do you have food during while watching TV? □ Yes □ No

11. How many meals do you have daily? □ 1 □ 2-3 □ more than 3

12. Do you have a school meals? □ Yes □ No

13. Do you exercising? □ Yes □ No

14. Do you play football? □ Yes □ No

15. Did you receive family health education on obesity? □ Yes □ No

If the answer is yes-----------------------

Determine where do get this education? ---------------------

16. Do you received breastfeeding? □ Normal □ artificial

Duration of breastfeeding? ------------------

Duration of artificial feeding? ------------------

17. Do you agree to withdraw a blood sample from your child? □ Agree □ Disagree

Agreement: I agree to complete this questionnaire concerning health of my children.

Signature: -----------------------------

Date: -----------------------------

Thank you for your cooperation

Researcher / Abeer Mohammed Siam
استبانه

استبانان لدراسة السمنة والعوامل المساعدة على انتشارها بين تلاميذ مدارس الابتدائية في غرب مدينة غزة

ولى أمر الطالب الفاضل/أرجو مساعدتك في إتمام هذه الدراسة (بحث ماجستير أحياء/جامعة الإسلامية) التي تختص بانتشار السمنة بين طلاب المرحلة الابتدائية في قطاع غزة، حيث أن هدفها تحديد العوامل المؤثرة في انتشارها، وذلك من خلال مساعدة أطفالكم في تعنيف الاستبانان واخذ عينة من أطفالكم في إجراء بعض الفحوصات عليها.

ملاحظة: أرجو قراءة فقرات الاستبانة بدقة ووضع إشارة X في المربع عند الإجابة المختارة

اسم الطالب ربعما:------------------------------------------
عنوان السكن:------------------------------------------
الصف:------------------------------------------
اسم المدرسة:------------------------------------------
التعليم:------------------------------------------
العمر:------------------------------------------

1. تعليم الأب: □ جامعي □ ثانوي □ إعدادي □ ابتدائي □ غير متعلم
2. تعليم الأم: □ جامعي □ ثانوي □ إعدادي □ ابتدائي □ غير متعلمة
3. وظيفة الأب: □ موظف حكومة □ موظف خاص □ عامل □ مقاعد
4. وظيفة الأم: □ موظفة حكومة □ موظفة خاص □ عاملة □ مقاعد
5. مستوى دخل الأسرة: □ أقل من 1000 شكل □ من 1000 إلى 1500 □ من 1500 إلى 2000 □ من 2000 إلى 2500 □ 2500 فما فوق
6. هل هناك أحد في العائلة يعاني من السمنة □ نعم □ لا
7. هل كانت الإجابة نعم أما إذا كانت الإجابة نعم ما درجة القرابة □ أب/أم □ أخ/أخت □ جد/جهة □ عمة/خالة
8. هل تشرب المشروبات الغازية؟ □ نعم □ لا
9. هل كانت الإجابة نعم إذا كانت الإجابة نعم
   مرة في الأسبوع □ 2-4 مرات في الأسبوع □ يوميا
   هل تتناول الوجبات الحلوة (متوكل، الكيك، الشوكولاتة) □ نعم □ لا
10. هل تتناول الطعام أثناء مشاهدة التلفاز؟ □ نعم □ لا
Agreement: I agree to complete this questionnaire concerning health of my children.

أنا موافق على تعبئة هذا الاستبيان الذي يتعلق بصحة أبنائي.

التوقع ولن الأمر: __________________
التاريخ: __________________

شكرًا لك على حسن تعاونك

الباحثة: عبير محمد صيام
Annex 2

Palestinian National Authority
Ministry of Health
Helsinki Committee

Name:

I would like to inform you that the committee has discussed your application about:
"Obesity among primary school male children aged 10-12 years in West Gaza City".

In its meeting on June 2010 and decided the Following:-

To approve the above mention research study.

Signature

Member

Member

Chairperson

Conditions:-

- Valid for 2 years from the date of approval to start.
- It is necessary to notify the committee in any change in the admitted study protocol.
- The committee appreciate receiving one copy of your final research when it is completed.
الموضوع: تسييل مهمة باحث

نفيت سيناءكم عنً بأن طالبة الماجستير الباحثة شير محمد صبرة ومتولمة في مدرسة
الصموء بن العاص (ب)، موضوع الدراسة:

السمات بين الأطفال الذكور في مدارس منطقة غرب غزة،

حيث أن الدراسة تهدف إلى تحديد بعض المؤثرات في الدائرة في المرحلة العمرية (10-12) سنة وتحديداً عوامل البالغ، ودبي علاقته في حدوث النسبة، وتسعى الدراسة من خلال فحص عينات تم تجميعهم في قانوني المدارس الأول والثاني، وباستخدام.

سُوف يتم تجميع البيانات بشراف طبي من قبل إدارة الصحة المدرسية بوزارة الصحة حيث يتم التسجيل.

مرجى تسييل مهمة الباحثة حيث أن الفئة المستهدفة هي طلاب الفصل الخامس والسادس الذكور من جمع مدارس مدارس غرب غزّة.

مع واجب الإخلاص،

د. زياد محمد شيرب

وكيل وزارة المساعد للمدارس في

التعليم والنشر،

تاريخ: 3-7-2010

السلطة الوطنية الفلسطينية
وزارة التربية والتعليم العالي
مكتب الرؤوس المساعد للمدارس التعليمية

الإدارة العامة للصحة المدرسية
الاسم: الدكتور محمد صبرة
التاريخ: 3-7-2010
الموقع: 17 أيار (ун) 1431 هـ

السيد مدير التربية والتعليم: غزّة
المالع على الله ورحمة الله وبركاته...